51. (New) The method of claim 47, wherein said tumor is a solid tumor or a hematological tumor.

52. (New) The method of claim 47, wherein said tumor is selected from the group consisting of melanoma, lymphoma, plasmacytoma, sarcoma, glioma, thymoma, leukemias, breast cancer, prostate cancer, colon cancer, esophageal cancer, brain cancer, lung cancer, ovarian cancer, cervical cancer and hepatoma.

53. (New) The method of claim 47, wherein said animal is a human subject.

REMARKS

I. General Remarks

Claim 45 is amended correct obvious typographical errors. In claims 26 and 41 the word transformed is replaced with the word transfected a term preferred by Applicant. Both terms mean the introduction of DNA into cells. This replacement does not introduce any new matter and is fully supported by the specification. For example, at page 48 Example 5 describes transfection of LM-IL-2K^b cells with genomic DNA from B16 melanoma cells.

II. Patentability Arguments

A. The Rejections Under 35 U.S.C. §112, Second Paragraph, Should Be Withdrawn

The Examiner has rejected claims 26, 41-46 under 35 U.S.C. § 112, second paragraph, as allegedly being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Specifically, the Examiner alleges that the term "effective amount" recited in claim 26 is not supported by the instant specification. Applicant respectfully brings to the attention of the Examiner that claim 26 defines the effective amount as the dosage sufficient for accomplishing a function of the inhibition or prevention of tumor growth and that the determination of such dosages is a matter of routine (see MPEP, page 2100-199). The court has ruled that the term "an

effective amount" is definite when a person skilled in the art can determine specific values for the amount based on the disclosure. In *re Mattison*, 509 F.2D 563, 184 USPQ 484 (CCPA 1975).

The instant specification teaches that the effective amount for treating cancer in each particular patient can be determined individually by a physician who makes such a determination by using the teachings of the specification and by taking in account the patient's weight, tumor size, extent of metastasis and general condition of the health.

The Examiner alleges that with regard to claim 26 and dependent claims thereof, it is not clear whether the tumor is present or absent prior to administering of the antigen-presenting cells. Claim 26, as currently amended, recites a method of treating a tumor. A person skilled in the relevant art would understand that tumor is present prior to administering of the antigen-presenting cells.

However, the instant invention also teaches methods of preventing tumor formation using tumor DNA not taken directly from the animal but from separate tumor cells. The methods of preventing tumor formation are now claimed in newly added claims 47, 48, 49, 50, 51, 52 and 53. In Example 6, at pages 50 through 53, applicant demonstrates that when lab animals are immunized with the semi-allogeneic cells prepared according to the present invention, the animals become resistant to later inoculation with a tumor.

The Examiner raises an issue as to how a person skilled in the relevant art would prepare a vaccine for which a tumor cell DNA is required before any tumor has developed in a patient. As shown in Example 6, a vaccine was prepared using DNA from tumor cells not taken from the animal. Thus it is readily apparent that based on the specification the vaccines may be prepared not only by using DNA from tumors taken from the animal, but also from other tumor DNA sources.

The Examiner also raises an issue that the inoculation with a tumor is not equal to a real life situation wherein there appears to be no inoculation with a tumor but rather the tumor reoccurs spontaneously. An experimental system in which laboratory animals are inoculated with a tumor is a standard system for evaluating whether or not tumors can be treated or prevented. This assertion is supported by Exhibit A, which includes two recent articles using such an experimental system for developing cancer prevention treatments.

With regard to Examiner's comment in paragraph 7 on page 2 of the Office Action, where the Examiner alleges that the terms "MHC I and MHC II determinants" are not adequately defined in the specification, applicant respectfully submits that the terms are well known terms of art. This assertion is supported by Exhibit B, which includes a copy of a recent abstract from a scientific paper, wherein the term "MHC determinants" is used. In summary, applicant submits that since the term "MHC determinant" is a well established term of art, a person of ordinary skill in the art will unequivocally understand the meaning of the term as it is used in the instant specification.

In paragraph 8 on page 3 of the Office Action, the Examiner alleges that the term "genomic DNA" of claim 26 is not adequately defined. Applicant respectfully submits that the term "genomic DNA" is a term well known to one of ordinary skill in the art. In support of this assertion, applicant submits Exhibit C, which includes description of a kit which can be used for genomic DNA purification. In addition, applicant brings to the attention of the Examiner that in a paragraph bridging pages 30 and 31, and in a paragraph thereafter, the instant specification describes genomic DNA isolated from tumor cells of a patient and also teaches how to isolate the DNA, transform cells with the DNA and screen for transfected cells expressing tumor antigens from the DNA. Furthermore, in Example 5 at page 48 through 49, applicant teaches in detail how to isolate genomic DNA from cancer cells and how to use this isolated DNA for transformation of LM-IL-2K^b cells. Moreover, applicant demonstrates in Examples 6 and 7 that cells transfected with genomic DNA, according to the teachings of Example 5, are suitable for both treating and preventing cancer.

In summary, applicant respectfully submits that amended claim 26 and its dependent claims particularly point out and distinctly claim the subject matter which applicant regards as the invention and therefore the rejection should be withdrawn.

B. The Rejections Under 35 U.S.C. § 112, First Paragraph, Should Be Withdrawn

Claims 26, 41-46 stand rejected under 35 U.S.C. § 112, first paragraph. At page 3 of the Office Action, beginning in paragraph 9, the Examiner alleges that the specification does not enable a person skilled in the art to prevent a tumor in a subject comprising the administration of an antigen-presenting cell expressing syngeneic MHC II molecules, allogeneic MHC II molecules, any and all cytokines, and genomic DNA from tumor.

The Examiner alleges that the specification is not enabling with respect to using MHC II determinant for the purposes of the instant invention. The Examiner has cited Roitt et al. Immunology 4th ed. 1998 ("Roitt") to support his knowledge about differences between class I and class II MHC molecules. Indeed, MHC I molecules are present on surfaces of virtually all nucleated cells and trigger $CD8^+$ -dependent cytolytic immune responses, while MHC II molecules are present on surfaces of only a subpopulation of cells (dendritic cells, B lymphocytes and macrophages) and trigger $CD4^+$ -dependent immune responses. However, either an MHC I triggered response or an MHC II triggered response would suit the purposes of the instant invention.

The instant specification teaches how to immunize a patient against his/her tumor cells with antigen-presenting cells prepared according to the present application. As stated at page 18 of the instant specification, the antigen-presenting cells may be either professional antigen-presenting cells or facultative antigen-presenting cells.

The specification then teaches, by the way of exemplification, how to transform antigen-presenting cells with MHC I determinant. A person skilled in the art is fully enabled by the instant specification to transform antigen-presenting cells with the MHC II determinant using the same method without undue experimentation by following the procedures described in the working examples for the MHC I determinant.

In summary, the instant specification fully enables a person skilled in the relevant art to practice the invention under conditions when syngeneic and allogeneic MHC II determinants are used for vaccine preparation.

The Examiner also alleges that the specification is devoid of teaching a method of preventing a tumor. As it was discussed *supra*, Example 6 of the instant specification demonstrates that immunization with semi-allogeneic antigen presenting cells prepared according to the specification is beneficial for preventing tumor development.

The Examiner also alleges that the instant specification does not enable a person skilled in the art to use genomic DNA of tumor cells for transfection of the antigen-presenting cells. As it was discussed *supra*, a person skilled in the art is fully enabled by the instant specification at page 30 through 32 and by Example 5 at pages 48 through 49 which gives working examples of obtaining tumor cells, isolating genomic DNA from the cells and then using the isolated genomic

DNA for transfection of antigen-presenting cells and then using these transfected cells to practice the instant invention.

The Examiner alleges that the instant specification does not enable a person skilled in the art to use different cytokines for the methods of the instant invention. Applicant respectfully submits that at page 33 through 35 of the instant specification, applicant provides a list of cytokines that can be used for the purposes of the instant invention. Applicant then teaches in detail how to use any of the cytokines for transformation of the antigen-presenting cells of the instant invention. Applicant then demonstrates by the way of non-limiting examples that IL-2 cytokine can be used for preparing anti-cancer vaccines by methods of the instant invention. A person skilled in the art is fully enabled by the instant specification to select any cytokine from a list provided by the instant specification and to construct the antigen-presenting cells transfected with the selected cytokine by following teachings of the instant specification for IL-2.

The Examiner also raises an issue that different cytokines trigger different activation pathways. The Examiner then alleges that because different cytokines trigger different activation pathways, the teachings for IL-2 may not be applicable to other cytokines. However, as it is explained in the instant specification, any type of antigen-presenting cell activation is sufficient for the purposes of the instant specification. Therefore, the fact that different cytokines function in different activation pathways is irrelevant to methods of the instant specification.

The Examiner raises the issue that IL-2 in large amounts may be toxic to a patient and then alleges that a vaccine containing transfected antigen-presenting cells expressing IL-2 may be toxic to a patient. However, the present specification demonstrates (by the way of working examples) that an IL-2 expressing vaccine made according to the present invention is beneficial for treating a cancer in a subject with no obvious toxic effect on the subject.

In summary, applicant respectfully submits that the instant specification fully enables a person skilled in the art to practice the instant invention as claimed and therefore, rejections under 35 U.S.C. § 112, first paragraph, should be reconsidered and withdrawn.

C. The Rejections Under 35 U.S.C. § 102 (b) and (e) Should Be Withdrawn

The Examiner has rejected claims 26, 41, 42 and 44-45 under 35 U.S.C. § 102(b) as allegedly being anticipated by Eisenbach et al. (EP 0 569 678 A2; Eisenbach). The Examiner

characterizes Eisenbach as teaching a method of treating tumorous diseases, which includes lung cancer, with a tumor cell expressing both a syngeneic and allogeneic MHC molecule.

The subject matter claimed by the instant specification is different from Eisenbach in a number of ways, for example Eisenbach does not teach transforming cells with tumor DNA to prepare a vaccine and as such can not properly anticipate the claims of the instant application.

The Examiner has also rejected claims 26, 41, 42 and 44-45 under 35 U.S.C. § 102(e) as allegedly being anticipated by Eisenbach et al. (U.S. 5,750,102; Eisenbach 2). The Examiner characterizes Eisenbach 2 as teaching a method of treating tumorous diseases, which include lung cancer, with a tumor cell expressing both a syngeneic and allogeneic MHC molecule. Teachings of Eisenbach 2 have the same deficiencies as teachings of Eisenbach because Eisenbach 2 does not teach transforming cells with tumor DNA and as such can not properly anticipate the claims of the instant application.

In summary, since recited art does not disclose all of the elements claimed in the instant invention, applicant respectfully submits that the 102(b) and 102(e) rejections are improper and should be reconsidered and withdrawn.

Conclusion

Applicant respectfully submits that the amended claims are in condition for allowance and early notification thereof is requested. If in the interest of expediting prosecution, the Examiner has questions or comments he is invited to telephone the undersigned at the indicated telephone number.

Respectfully submitted,

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Research

Inhibitory effect of suberanilohydroxamic acid, a histone deacetylase inhibitor, on NMU-induced mammary tumorigenesis.

Suberoylanilide hydroxamic acid (SAHA), a histone deacetylase inhibitor, has been shown to inhibit the development of N-methylnitrosourea (NMU)-induced rat mammary tumors when fed in the diet continuously for the duration of the carcinogenic process. The present study was designed to determine whether the inhibitory effects of SAHA occur during the initiation process or at subsequent stages in the carcinogenic process. In addition, animals with established NMU tumors were administered SAHA to determine whether SAHA could inhibit the continued growth of established mammary tumors. It was found that SAHA fed at 900 ppm in the diet inhibited tumor yields when administered from 14 days prior to NMU administration to termination (-14 to +130) and from +14 and +28 days to termination. However, SAHA had no effect on tumor yields when administered from -14 to +14 or from -14 to +50 days and then returned to the control diets for the remainder of the experimental period (130 days). These results indicate that the inhibitory effects of SAHA are not exerted at the initiation phase of NMU-induced mammary tumorigenesis and appear, instead, to inhibit the subsequent stages in tumor development. Of most interest was the ability of SAHA to inhibit the growth of established mammary tumor. Administration of SAHA in the diet at 900 ppm resulted in significant inhibition of established tumor growth. Thirty-two percent of SAHA-treated tumors exhibited partial regression compared to 12% of controls, growth was stabilized in 24% of treated tumors compared to 12% of controls while 11% exhibited complete regression compared to 0% of controls. Collectively, SAHA-treated tumors exhibited a 7-fold reduction in growth compared to untreated tumors over the test period. The results of this animal model study indicate that SAHA, when fed in the diet, serves as both a chemopreventive and chemotherapeutic agent in the absence of any detectable side effects. Anti Cancer Res. 22:1497-1504, 2002.

Effect of soy protein isolate and conjugated linoleic acid on the growth of Dunning R-3327-AT-1 rat prostate tumors

The objective of this study was to test the hypothesis that supplementation of the diet with combinations of isoflavone-rich soy protein isolate (SPI) and conjugated linoleic acid (CLA) would act to inhibit the growth of androgen-independent R-3327-AT-1 rat prostate tumor cells inoculated ectopically into male Copenhagen rats. The results of this study indicate that neither SPI or CLA inhibit the in vivo growth of prostate tumors when administered either singly or in combination. Moreover at the highest concentrations i.e. 20% and 10% SPI there was a statistically significant increase in tumor volume compared to unsupplemented controls. These results in an established rat model suggest caution in the use of soy isoflavone-rich soy supplements in human studies involving hormone-refractory prostate cancer. The Prostate (In Press Dec/Jan 2002-2003).

Clinical and intervention trial: Longitudinal study of the effects of soy isoflavones on indicators of breast cancer risk in healthy premenopausal women.

The objective of this study was to assess the effects of a soy isoflavone supplement on urinary 20HE1/16a OHE1 ratios after 1 month administration of 120 mgs isoflavones/day. Thirty-six women were recruited at St. Lukes-Roosevelt Hospital Center, demographics obtained by interviewer and a self-administered food frequency questionnaire recorded. Blood and urine samples were collected 3 times, at baseline, after 1 month on soy and after one month off soy, urinary isoflavones were measured by HPLC methods to assure compliance and estrogen metabolite ratios were determined by solid phase competitive immunoassay. It was found that all but 3 women exhibited increased isoflavone excretion following soy intake which returned to low or undetectable levels following return to customary diet. Thirteen women exhibited decreased, and 20 women exhibited increased ratios. There was no association between increased or decreased ratios and ethnicity, BMI, alcohol intake, fiber intake, smoking behavior or activity levels. Those women with increased ratios exhibited no clear cut pattern with regard to which metabolite increased or decreased. While some exhibited increased 20HE1 others showed a decrease, in both metabolites with 16a OHE1 decreasing to a greater extent. The results of this study do not support the hypothesis that soy intake alters the 20HE1/16a OHE1 ratio towards a low-risk profile. On going studies on peripheral blood lymphocytes obtained from these subjects indicate that cytochrome 1B1 which is involved in steroid metabolism and, in particular, the 40 HE1 pathway, is suppressed during soy consumption and returns to pre-supplement levels after return to the customary diet. The result of this study, done in conjunction with Dr. Peter Holt of the IFCP, and Drs. L. Bradlow and D. Sepkovic of the Institute Biomedical Research (Hackensack, NJ), will be presented at the 94th Annual Meeting of the AACR in Toronto, Canada, April 5-9, 2003.

Scientific Activities

A review of animal model studies of tomato carotenoids, lycopene and cancer chemoprevention.

There are relatively few reports on the cancer chemopreventive effects of lycopene or tomato carotenoids in animal models. The majority, but not all, of these studies indicate a protective effect. Inhibitory effects were reported in two studies using aberrant crypt foci, an intermediate lesion leading to colon cancer, as an endpoint and in two mammary tumor studies, one using the dimethylbenz(*a*)anthracene model, and the other the spontaneous mouse model. Inhibitory effects were also reported in mouse lung and rat hepatocarcinoma and bladder cancer models. However, a report from the author's laboratory found no effect in the N-nitrosomethylurea-induced mammary tumor model when crystalline lycopene or a lycopene-rich tomato carotenoid oleoresin was administered in the diet. Unfortunately, because of differences in routes of administration (gavage, intraperitoneal injection, intra-rectal instillation, drinking water, and diet supplementation), species and strain differences, form of lycopene (pure crystalline, beadlet, mixed carotenoid suspension), varying diets (grain-based, casein based) and dose ranges (0.5-500 ppm), no two studies are comparable. It is clear that the majority of ingested lycopene is excreted in the feces and that 1000-fold more lycopene is absorbed and stored in the liver than accumulates in other target organs. Nonetheless, physiologically significant (nanogram) levels of lycopene are assimilated by key organs such as breast, prostate, lung, and colon, and there is a rough dose-response relationship between lycopene intake and blood levels. Pure lycopene was absorbed less efficiently than the lycopene-rich tomato carotenoid oleoresin and blood levels of lycopene in rats fed a grain-based diet were consistently lower than those in rats fed lycopene in a casein-based diet. The latter suggests that the matrix in which lycopene is incorporated is an important determinant of lycopene uptake. A number of issues remain to be resolved before any definitive conclusions can be drawn concerning the anticancer effects of lycopene. These include the following: the optimal dose and form of lycopene, interactions among lycopene and other carotenoids and fat soluble vitamins such as vitamin E and D, the role of dietary fat in regulating lycopene uptake and disposition, organ and tissue specificity, and the problem of extrapolation from rodent models to human populations. Soc. Exp Biol Med 227:864-868, 2002

International Symposium on the Role of Tomato Products and Carotenoids in Disease Prevention

Dr Cohen organised a symposium, entitled "International Symposium on the Role of Tomato Products and Carotenoids in Disease Prevention" was held at the New York Academy of Medicine, in April 2001. The Symposium consisted of 13 speakers covering topics ranging from the chemistry and metabolism of lycopene to clinical intervention studies. Studies using mixtures of tomato carotenoids effects of tomato carotenoids on animal models of cancer, biomarkers of coronary artery disease, and ozone-induced lung dysfunction, were also reviewed. A poster session was held after the meeting during which 18 studies were presented. The proceedings of the Symposium have been published in the November 2002 issue of Society Experimental Biology & Medicine (SEBM). This is the 2nd Symposium on this topic organized by the IFCP. The first was held on March 1997 and the proceedings published in the June 1998 issue of the SEBM.

Nutrition and Cancer

Dr. L.A. Cohen is the editor of Nutrition and Cancer, a peer reviewed journal, which is published six times a year. The journal which has been in existence for 30 years, publishes original research and review articles covering the field of nutritional carcinogenesis including epidemiology, clinical intervention, animal model studies, in vitro and molecular studies. The journal is ranked among the top 10 nutrition journals in the US.

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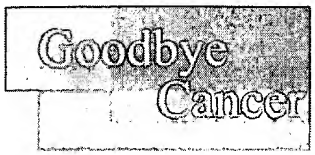

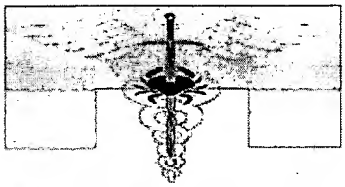

GENERAL CONCLUSIONS

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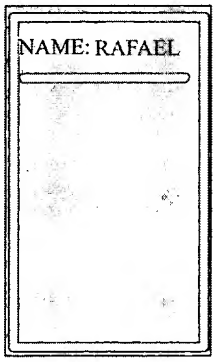

The definitive result reported here show that the studied substance termed LYMPHOTONIC PF-2 clearly has anti-tumor not only due to the direct Cytotoxic or cytostatic action, but also due to the activation of the defence system of the organism in general. It was shown by its ability to prevent cancer in the animal model where LYMPHOTONIC PF-2 proved to be an effective prophylactic agent. This property of LYMPHOTONIC PF-2 allows us to call it an immunomodulator. Further studies are planned in order to investigate the anti-tumor activity of LYMPHOTONIC PF-2 in the radiation-induced and carcinogen-induced tumor models. Its low toxicity and high anti-tumor activity in the experimental animals makes LYMPHOTONIC PF-2 a perspective candidate for its use in a phase I of the pre-clinical trial for the investigation of its possible use in the cancer therapy.

Summarizing all experimental data, reviewed in this manuscript, following conclusions can be drawn:

1) LYMPHOTONIC PF-2 has no mitogenic effect on the human PBLs in vitro.

2) LT does not induce the secretion of the cytotoxic factors by human PBLs in-vitro.

3) LYMPHOTONIC PF-2 does not change the anti-tumor activity of anti-tumor drugs (i.e.doxorubicin).

4) LYMPHOTONIC PF-2 no reversible effect on the multiple-drugs resistance of cancer cells.

5) LYMPHOTONIC PF-2 increases the cytotoxic activity of the peripheral blood lymphocytes, monocytes and neutrophils of healthy donors and cancer patients against malignant target cells in vitro.

6) High concentration of LYMPHOTONIC PF-2 are cytotoxic against rapidly proliferating normal and malignant cells.

7) LYMPHOTONIC PF-2 induces the increase of cytotoxic activity of immunocompetent cells against malignant cells both in vitro and in vivo.

8) Fractionation of LYMPHOTONIC PF-2 and analysis of the fractions showed that the stimulating effect of LYMPHOTONIC PF-2 on cytotoxic activity of PBLs is determined by the multi-component mixture of lypophilic substances, extracted by hexane and chloroform

and also by the water-soluble high molecular, carbohydrate-containing fraction.

9) LYMPHOTONIC PF-2 is non-sterile, contaminated by bacteria and fungi, that makes it impossible to store without sterilization by filtration, though this procedure leads to the partial loss of its biologic activity.

10) Studies carried out in the animal model on mice with tumors, induced by inoculation of the autologous tumor cells showed that LYMPHOTONIC PF-2 significantly reduces frequency of tumor formation, and when used in high doses, increases the mean life span of the treated animals. LYMPHOTONIC PF-2 inhibits tumor growth and appeared to be very effective in the prevention of tumor formation, so it can be used for the prophylaxic cancer.

Definition Trials Prevention Conclusion References Scientific Team Publications How to Order
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1: Hum Immunol. 2000 Dec;61(12):1352-62.

ELSEVIER FULL-TEXT ARTICLE

Induction of T-cell response to cryptic MHC determinants during allograft rejection.

Boisgerault F, Anosova NG, Tam RC, Illigens BM, Fedoseyeva EV, Benichou G.

Cellular and Molecular Immunology Laboratory, Schepens Eye Research Institute, Harvard Medical School, Boston, MA 02114, USA.

The presentation of MHC peptides by recipient and donor antigen presenting cells is an essential element in allorecognition and allograft rejection. MHC proteins contains two sets of determinants: the dominant determinants that are efficiently processed and presented to T cells, and the cryptic determinants that are not presented sufficiently enough to induce T-cell responses in vivo. In transplanted mice, initial T-cell response to MHC peptides is consistently limited to a single or a few immunodominant determinants on donor MHC molecule. However, in this article we show that under appropriate circumstances the hierarchy of determinants on MHC molecules can be disrupted. First, we observed that gamma IFN can trigger de novo presentation of cryptic self-MHC peptides by spleen cells. Moreover, we showed that allotransplantation is associated with induction of T-cell responses to formerly cryptic determinants on both syngeneic and allogeneic MHC molecules. Our results suggest that cross-reactivity and inflammation are responsible for the initiation of these auto- and alloimmune responses after transplantation.

PMID: 11163093 [PubMed - indexed for MEDLINE]

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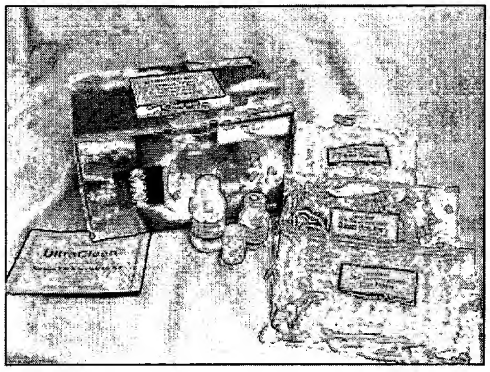

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Features and Benefits

- Format: silica spin filter method, 20 minute protocol
- Yields DNA greater than 50 kb in length from tissue
- DNA is ready for PCR, restriction digests, all down stream applications
- Kits include all reagents ready to use, all required spin filters and tubes
- User friendly

Description of method
Use this kit to isolate genomic DNA from muscle, hair, bone and organ tissue. Without the use of organic solvents like phenol and chloroform, this safe kit will isolated genomic DNA from tissue. Fresh or frozen tissue samples (25mg) are added to a bead beating tube. This tube is briefly vortexed on an inexpensive vortex adapter (available from Mo Bio Laboratories, Inc. Catalog number: 13000-V1). The cells are lysed and the released DNA is bound to a silica spin filter. The filter is washed, and finally the DNA is released into guaranteed DNA free, 10mM Tris.

Protocols:
- Word Doc

Kit Contents:
- 2 ml Bead Solution tubes (contains 550ul solution) 250
- Solution TD1.... 275 ml
- Solution TD2.... 4x32 ml
- Solution TD3.... 14 ml
- Spin filters units in 2.0 ml tubes (250)
- Collection tubes 2.0 ml (750)

Specifications
Yield: Up to 40 µg
Time: 20 minutes
DNA quality: A260/A280=1.8-2.0

MSDS:
- Word Doc

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